Early childhood risk factors for sensitization at school age

Joachim Kuehr, MD,[a] Thomas Frischer, MD,[a] Wilfried Karmaus, MD, MPH,[b] Rolf Meinert, MSc,[a] Regina Barth, MD,[a] Edelgard Herrman-Kunz, MD,[a] Johannes Forster, MD,[a] and Radvan Urbanek, MD[c]

Freiburg and Hamburg, Germany, and Vienna, Austria

Early childhood risk factors for current sensitization were investigated by use of cross-sectional data of a longitudinal study in Southwest Germany. Information was gathered by questionnaires from 1812 families of whom 1470 children 6 to 8 years old were tested by means of a skin prick test (SPT) with seven aeroallergens. Groups with sensitization (n = 201: positive SPT to grass pollens 6.6%, Dermatophagoides pteronyssinus 6.5%, Dermatophagoides farinae 4.4%, cat dander 4.6%, any of the tested allergens 13.7%) are compared with children without sensitization (n = 1269). As risk factors for any sensitization parental atopy (odds ratio [OR]/95% confidence interval [95%CI]: unilateral 1.9/1.3 to 2.6; bilateral 2.8/1.5 to 5.2), low gestational age (1.9/1.1 to 3.2), and male gender (1.6/1.2 to 2.3) are statistically significant in multiple logistic regression. Former cat ownership is significantly related to sensitization to cat dander (2.7/1.4 to 5.5). Breast feeding, maternal smoking habits after the child's birth, prior exposure to pets, and social class are not important. In conclusion, our data suggest parental atopy, low gestational age, and male gender as independent risk factors for sensitization to aeroallergens at school age. (J ALLERGY CLIN IMMUNOL 1992;90: 358-63.)

Key words: *Childhood, risk, sensitization, familial, smoking, gender*

In childhood, sensitization to aeroallergens is a major cause for chronic respiratory diseases such as hay fever and bronchial asthma.[1-3] Numerous epidemiologic and clinical studies were carried out to analyze the pathogenesis of atopic diseases.[4-14] Sensitization to allergens is usually estimated by use of skin prick tests (SPT) or by measurement of allergen-specific IgE antibodies.[4-7, 9, 10, 13, 14] Some studies have demonstrated that risk factors for atopy (i.e., allergen exposure) may act antigen specific.[5, 6, 13-16] Parental atopy (PA) and early exposure to potent allergens like cow's milk proteins or pollens are risk factors for sensitization in later life.[4, 6, 11-13, 16-19] Presumable risk factors for sensitization are social class,[20-22] gender,[10, 23-25] early respiratory infections,[5, 7, 26, 27] and tobacco smoke exposure.[8, 12, 14, 28, 29]

Abbreviations used

SPT:	Skin prick test
PA:	Parental atopy
LGA:	Low gestational age
MSP:	Maternal smoking during pregnancy
Dpt:	*Dermatophagoides pteronyssinus*
Df:	*Dermatophagoides farinae*
OR:	Odds ratio
95% CI:	95% confidence interval
HDM:	House dust mite

We analyzed data derived from a cross-sectional investigation of an ongoing longitudinal study in Southwest Germany comprising 1812 schoolchildren to test these risk factors in regard to sensitization in SPT. In addition, we investigated low gestational age (LGA) and potential combined effects on current sensitization to aeroallergens.

POPULATION IN THE STUDY AND METHODS

The data were collected in a first cross section of a longitudinal study designed to assess risk factors for allergy

From the University Children's Hospital, Freiburg,[a] NORDIG Institute for Health Research and Prevention, Hamburg,[b] and University Children's Hospital, Vienna.[c]

Supported by the German Federal Ministry for Research and Technology.

Received for publication Nov. 19, 1991.

Revised April 21, 1992.

Accepted for publication April 30, 1992.

Reprint requests: Joachim Kuehr, MD, University Children's Hospital, Mathildenstr. 1, W-7800 Freiburg, Germany.

1/1/39065

9002

2024228028

Source: https://www.industrydocuments.ucsf.edu/docs/ppvj0000

and asthma in children. To obtain a population-based sample of schoolchildren between 6 and 8 years of age, all parents whose children had started elementary school were invited to participate. Questionnaires were distributed to 2604 families living in three urban areas in Southwest Germany (Freiburg, Kehl, and Lörrach) from February to April 1990. The questionnaires were returned by 1812 families (participation: 70%). In 94% the questions were answered by the mothers. Appended to the questionnaire was a written consent for the children to participate in further investigations (SPT and free running test). The SPT took place from March to June in the same year. A population of 1470 children participated in the SPT. The study was approved by the local ethical committee and also by local school authorities.

Questionnaire

Thirty to 45 minutes were used to fill in answers. The reproducibility of the answers was determined in a separate study with 162 primary school children. Questionnaires were administered by the parents on two occasions 14 days apart from each other. In the following the proportion of agreement (%) is given in brackets. For the analyses, questions of interest are the following: parental atopy (PA, maternal or paternal history of asthma or hay fever or eczema; 94%); low gestational age (LGA, child born at least 3 weeks preterm); maternal history of cigarette smoking during pregnancy (MSP, 80%), in the first year of the child's life (81%), and in the year preceding the cross-sectional investigation (85%); duration of breast feeding (in months); history of pneumonia and history of whooping cough in the child's first year of life (94% and 87%); history of an exposure to pet animals at home (previous exposure and current exposure); social class (highest educational level achieved at school by father or mother: 87%); gender of the child.

SPT procedure

Seven purified and immunochemically characterized aeroallergens (Allergologisk Laboratorium, A/S, Copenhagen, Denmark) were applied to the child's forearm with the aid of an uncoated Phazet needle (Pharmacia Diagnostics, AB, Uppsala, Sweden). The reactions were recorded after 15 minutes.[30] Extracts from grass pollens (mixture of the six most common species), birch and hazel pollens, *Dermatophagoides pteronyssinus (Dpt)* and *Dermatophagoides farinae (Df)* as well as cat and dog dander were used (concentration: 10 histamine equivalent in prick testing). As control solutions, histamine hydrochloride (10 mg/ml) and sodium chloride (9 gm/L) were applied. The largest and the orthogonal diameter of a wheal were measured by means of a transparent ruler, and the arithmetic mean was calculated. A reaction to the negative control was subtracted from the allergen value. A wheal of 3 mm or more was classified as a positive reaction. The tests were carried out by two nurses under supervision of a physician.

Statistical analysis

The questionnaire data were categorized, and differences between the groups with and without reaction in SPT were examined by chi-square tests. The estimation of the relative risks was based on logistic regressions and provided odds ratios (ORs) and their 95% confidence intervals (95% CI).[31, 32] In the assessment we followed the backward procedure by Greenberg et al.[33] that starts with a model including all potential confounders. Reducing the model backward, the potential confounder social class could be eliminated if no change in the parameter estimates of the variables of interest occurred (≤10% in change). In addition, to investigate an effect of parental atopy together with MSP, gender, and LGA, ORs for the different combinations of these variables were calculated.[34] The expected ORs (OR_{exp}) for a combination of two factors were derived from observed ORs (OR_{obs}) as follows: $OR_{exp11} = OR_{obs10} + OR_{obs01} - 1$.[34] The formula is based on an additive model with no interaction.

RESULTS

A sensitization to any of the seven tested allergens is found in 13.7% of the 1470 children, of whom 6.6% are sensitized to grass pollens, 6.5% to *Dpt*, 4.4% to *Df*, and 4.6% to cat dander (Table I). In 109 children (7.4% of 1470) an SPT reaction occurs to at least one of the two house dust mites (HDMs) (46% to both, 41% to only *Dpt* and 13% to only *Df*).

Bivariate analyses

It is evident from Table I that unilateral PA (one atopic parent) is more frequent in children sensitized to any of the seven or to one of the single allergens tested, respectively. This is also true for the bilateral parental affection. Maternal atopy was reported in 21% compared with a paternal history in 18%. A gestational age of 37 weeks or less (LGA) is found in 7% of the nonatopic children and has a significantly higher prevalence in three of the sensitized subgroups (Table I). MSP is most frequent in the subgroup with no sensitization and is less frequent in children with sensitization to any allergen, *Df*, and cat dander. Former cat ownership is associated with a higher prevalence of sensitization to cat dander. In each of the sensitized subgroups more boys than girls are found. A history of pneumonia in the child's first year of life is rare and is reported significantly more frequently only in the group sensitized to cat dander.

Multivariate analyses

The potential effects of the variables of interest (listed in Table I) on the SPT sensitization were estimated by multiple logistic regression (Table II). The models were based each on the comparison of the sensitized subgroup ('any,' grass pollens, *Dpt*, *Df*, cat dander) with the nonsensitized population (n = 1122; missing data excluded). The ORs derived from the five regression models indicate a 1.6-fold to 2.5-fold risk of unilateral and a 1.5-fold to 3.1-fold risk of bilateral PA for sensitization. LGA is significantly related to sensitization to *Df* and *Dpt* as well as to any

2024228029

Source: https://www.industrydocuments.ucsf.edu/docs/ppvj0000

TABLE I. Percentages of risk factors for different SPT results

Factor	Sensitization to: None (n = 1269)	Any (n = 201)	Grass (n = 97)	Dpt (n = 95)	Df (n = 64)	Cat (n = 67)
Parental atopy						
None	67.3	51.2***	52.6***	50.5**	50.0**	46.3***
Unilateral	28.5	39.3	36.1	41.1	39.1	49.3
Bilateral	4.2	9.5	11.3	8.4	10.9	4.5
Low gestational age	7.0	11.6*	7.2	18.1***	18.8***	12.1
Breast feeding						
<8 months	18.5	22.5	19.6	28.7	20.3	20.9
≤4 months <8	18.5	21.0	18.6	18.1	29.7	26.9
≤1 months <4	37.0	31.5	33.0	29.8	32.8	28.4
month <1	25.9	25.0	28.9	23.4	17.2	23.9
Maternal smoking						
Pregnancy	15.8	9.1*	8.6	10.6	3.2**	6.1*
First year of life	23.5	18.1	17.9	20.2	15.9	16.7
Previous year	30.7	25.0	26.3	26.1	25.4	22.7
Whooping cough in the first year	6.0	5.6	6.4	4.4	3.3	7.7
Prior exposure to cat dander	9.6	11.0	9.3	7.4	7.8	19.4**
Current exposure to cat dander	16.6	10.5*	13.4	10.5	9.4	19.4
Prior exposure to pets	27.8	31.3	28.9	26.3	29.7	40.3*
Current exposure to pets	44.4	42.8	46.4	44.2	39.1	50.8
Social class						
Low	34.4	29.6	28.0	33.3	26.6	27.3
Medium	28.0	30.6	33.3	32.3	28.1	39.4
High	37.7	39.8	38.7	34.4	45.3	33.3
Gender (male)	46.8	60.2***	53.6	66.3***	64.1**	56.7
Pneumonia in the first year	2.4	4.0	2.1	5.3	3.1	9.0***

Percentages based on numbers after "missings" have been deleted.
Significance in chi-square test (*$p \leq 0.05$, **$p \leq 0.01$, ***$p \leq 0.001$) for the atopic groups compared with the group with no sensitization.

TABLE II. Odds ratios/95% confidence intervals of risk factors for atopy with mutual adjustment

Factor	Sensitization to: Any of 7 antigens	Grass pollens	Dpt	Df	Cat dander
Parental atopy					
Unilateral	1.87/1.32 - 2.63	1.64/1.02 - 2.64	2.23/1.37 - 3.62	1.70/0.95 - 3.04	2.50/1.44 - 4.33
Bilateral	2.79/1.51 - 5.18	3.13/1.42 - 6.92	2.76/1.16 - 6.55	2.98/1.17 - 7.55	1.52/0.43 - 5.42
Low gestational age	1.92/1.15 - 3.21	1.06/0.46 - 2.42	3.57/1.93 - 6.61	3.61/1.75 - 7.43	2.40/1.05 - 5.49
Maternal smoking in pregnancy	0.57/0.28 - 1.14	0.39/0.15 - 1.02	0.81/0.31 - 2.08	0.17/0.03 - 0.82	0.31/0.09 - 1.07
Prior exposure to cat dander	—	—	—	—	2.75/1.37 - 5.52
Gender male	1.64/1.19 - 2.26	1.21/0.78 - 1.89	2.12/1.33 - 3.39	2.20/1.26 - 3.84	1.68/0.99 - 2.86

Outcome variables: sensitization in SPT to 'any' allergen (n = 182), grass pollen (n = 87), *Dpt* (n = 86), *df* (n = 60), cat dander (n = 62), each in relation to no sensitization (n = 1122).

20242280030

Source: https://www.industrydocuments.ucsf.edu/docs/ppvj0000

allergen, with minor risk for the latter. With the exception of sensitization to *Df*, no significant relationship can be shown between SPT results and maternal smoking habits. In case of sensitization to *Df* MSP is less frequent than in the group with no reaction (OR, 0.17). In the children sensitized to cat dander former cat ownership is of significance for a current sensitization (OR, 2.75). Male gender is a significant risk for sensitization to both HDMs (*Dpt* and *Df*) and to any of the tested allergens.

Combined effects

As shown in Table II PA is a general predictor for sensitization in SPT. To investigate combined effects in which PA as one predictor might be involved, we analyzed combined effects with MSP, gender, and LGA, respectively, on SPT. For PA with gender and LGA no interaction could be seen (not shown). For MSP alone, a decreased risk of MSP in the absence of PA is found (ORs, 0.5 to 0.6; Table III). To consider different patterns of the mother's or father's atopic history and MSP, PA was additionally stratified for exclusive maternal or paternal history (bilateral parental atopy excluded). The ORs for maternal and paternal atopy indicate increased risks of both parental histories. A uniform combined effect of maternal and paternal atopy, respectively, together with MSP cannot be seen (ORs, 0.6 to 1.4). However, each of the observed ORs for the combinations are below the expected values. This pattern is not of significance, because the confidence intervals of the observed ORs include the expected values.

DISCUSSION

In the present epidemiologic study, PA, LGA, and male gender were identified as risk factors for allergic sensitization at school age.

To our knowledge, no previous results regarding the relationship between LGA and atopy in school-aged children are available. However, on the basis of the immune response of fetal and newborn mice, immunologic and digestive immaturity have been postulated to prevent tolerance in the case of an antigen feed in the neonatal period.[35] A high risk of early exposure to cows' milk for developing allergic reactions in early infancy has been shown in preterm babies with an additional family history of atopy.[36] This may be one potential explanation for the association of LGA with sensitization to common aeroallergens. However, the issue is still in dispute, whether such individuals with milk protein allergy have a higher likelihood of developing inhalative allergies later in life than do children without previous sensitization to food allergens. A sequence of this type is known in principle from the natural course of atopic diseases.[9] On the basis of our data we were not able to elucidate the sequence of these early events.

The role of gender in atopic diseases has repeatedly been reported. Several authors confirm our findings, which indicate that males have a stronger predisposition for sensitization than females.[10, 23, 24] However, a recently published large study did not find a relationship.[25] One explanation for a gender predominance may be the so-called Carter effect, assuming different thresholds of polygenic inheritance.[37] In addition to allergy related data, this hypothesis is supported by the predominance of males with bronchial asthma at school age.[38, 39] Male predominance in HDM allergy as well as bronchial asthma might be related to each other, considering that HDM allergens are regarded as the most important trigger of perennial allergic asthma in childhood.[40] Male gender and LGA have in common that an association with sensitization can be observed to occur most often in positive SPT to both types of HDM and less often with respect to at least one allergen (any) but not to cat dander and grass pollen (Table II). Therefore it can be assumed, that these risks may exist not only for atopy in general, but, as is the case here, for a specific phenotype.

Most authors have reported an effect of atopic family history that is comparable with our results.[12, 13, 18, 41] For children without genetic disposition, the prevalence of atopy varied between 12% and 27% compared with 10.8% in our population.[12, 41] In case of a positive family history the prevalences ranged between 23% and 43% compared with 19.1% in the current study.[12, 41] Findings vary according to country, study population, and instruments used. The results concerning the genetic effect on the offspring also appears to depend on the prevalence of atopy at each respective age. The effect of PA is estimated higher in children reaching school age than for infancy.[11-13, 18, 41, 42]

A differentiation of parental histories shows increased ORs for maternal (ORs, 1.5 to 2.3) as well as paternal atopy (ORs, 1.9 to 2.9) if the mother did not smoke during pregnancy (Table III). We did not expect that MSP as a single predictor would be associated with a lower frequency of sensitizations (ORs, 0.5 to 0.6). Hence, the combined risks of MSP and maternal (ORs, 0.7 to 1.4) as well as paternal atopy (ORs, 0.4 to 1.0) are not significant for sensitization. Although not significant, the observed negative association of MSP and sensitization in SPT was surprising to us.

An effect of MSP on fetal Ig regulation has been shown. In cord blood significantly higher levels of IgA, IgG, as well as IgM were found in the offspring

20242280031

Source: https://www.industrydocuments.ucsf.edu/docs/ppvj0000

TABLE III. Frequencies, OR, and 95% CI for combinations of PA with MSP

			Sensitization to								
			Any			Grass			Dpt		
Factor 1		Factor 2	Yes	No	OR/95% CI	Yes	No	OR/95% CI	Yes	No	OR/95% CI
PA	−	MSP −	90	688	1	43	688	1	42	688	1
PA	−	MSP +	10	139	0.5/0.3 - 1.1	5	139	0.6/0.2 - 1.5	5	139	0.6/0.2 - 1.5
Maternal	+	MSP −	39	168	1.8/1.2 - 2.7	16	168	1.5/0.8 - 2.8	18	168	1.8/1.0 - 3.1
Maternal	+	MSP +	3	24	1.0/0.3 - 3.2 (1.3)†	1	24	0.7/0.1 - 5.0 (1.1)	2	24	1.4/0.3 - 6.0 (1.4)†
Paternal	+	MSP −	34	134	1.9/1.3 - 3.0	17	134	2.0/1.1 - 3.7	18	134	2.2/1.2 - 3.9
Paternal	+	MSP +	2	25	0.6/0.1 - 2.6 (1.4)†	1*	25	0.6/0.1 - 4.8 (1.6)	1	25	0.7/0.1 - 5.0 (1.8)†

*To estimate the odds-ratio one artificial case was added to an empty cell, which gives conservative estimates.
†The values in brackets are the expected ORs for the combination of MSP with maternal and paternal atopy, respectively.

163 women who smoked, as compared with 130 women without a history of smoking.[43] After an 18-month follow-up, a significantly higher cord blood IgE in the case of maternal smoking was reported.[44] Smoking behavior, however, was established for the age of 18 months, but not for the pregnancy.[8] An effect of MSP on the cord blood IgE concentration could not be shown in a population of 136 newborn children, 16 of whose mothers had smoked during pregnancy.[45]

On the basis of the literature, (an) immunologic effect(s) of passive smoke exposure seem(s) likely. Nevertheless, a potential role of MSP in the atopic history of the offspring is still not sufficiently understood. Thus we do not interpret our results to show an independent effect of MSP on atopy in the offspring.

In regard to our findings we must point out that the retrospective ascertainment, which encompasses a period of time spanning 8 years, can be limiting for the reliability of risk assessment. Since the risk of LGA can have implications for the care of newborns, we examined the child's birth record in a representative sample of the population (n = 287). In 96%, consistency of questionnaire and birth record is found, and for 73% of children classified to LGA, accuracy of recall is demonstrated. The present analyses did not identify statistically significant relationships between breast feeding, postpartal maternal smoking, whooping cough in the first year of life, and educational status on one hand and sensitization in SPT on the other.

In conclusion, our data show that parental atopy represents a general risk for sensitization at school age. A higher risk of sensitization is seen in children with bilateral parental atopy, than in the case of unilateral parental atopy. Among the other variables of interest, LGA and male gender point to a significant relationship primarily to sensitization to HDM allergens. Furthermore, the observed significance of prior, but not of present, cat ownership in relationship to current sensitization provides one example that a recognition of changed patterns of behavior is necessary for retrospective ascertainments in epidemiologic studies.

We thank Mrs. Monika Mortensen and Mrs. Sabine Linser-Haar for their excellent collaboration in the field study conducting the SPT.

REFERENCES

1. Burrows B, Lebowitz MD, Barbee RA. Respiratory disorders and allergy skin-test reactions. Ann Intern Med 1976;84:134-9.
2. Haahtela T, Heiskala M, Suoniemi I. Allergic disorders and immediate skin reactivity in Finnish adolescents. Allergy 1980;35:433-41.
3. Peat JK, Britton WJ, Salome CM, Woolcock AJ. Bronchial hyperresponsiveness in two populations of Australian school children. II. Relative importance of associated factors. Clin Allergy 1987;17:283-90.
4. Kjellman NIM, Johansson SGO. IgE and atopic allergy in newborns and infants with a family history of atopic disease. Acta Paediatr Scand 1976;65:601-7.
5. Frick OL, German DF, Mills J. Development of allergy in children. I. Association with virus infections. J ALLERGY CLIN IMMUNOL 1979;63:228-41.
6. Croner S, Kjellman NIM, Eriksson B, Roth A. IgE screening in 1701 newborn infants and the development of atopic disease during infancy. Arch Dis Child 1982;57:364-8.
7. Weiss ST, Tager IB, Munoz A, Speizer FE. The relationship of respiratory infections in early childhood to the occurrence of increased levels of bronchial responsiveness and atopy. Am Rev Respir Dis 1985;131:573-8.
8. Magnusson CG. Maternal smoking influences cord serum IgE and IgD levels and increases the risk for subsequent infant allergy. J ALLERGY CLIN IMMUNOL 1986;78:898-904.
9. Cogswell JJ, Mitchell EB, Alexander J. Parental smoking, breast feeding, and respiratory infection in development of allergic diseases. Arch Dis Child 1987;62:338-44.
10. Astarita C, Harris RJ, de Fusco R, et al. An epidemiological study of atopy in children. Clin Allergy 1988;18:341-50.
11. Åberg N, Engström I, Lindberg U. Allergic diseases in Swedish school children. Acta Paediatr Scand 1989;78:246-51.

2024228032

Source: https://www.industrydocuments.ucsf.edu/docs/ppvj0000

Sensitization to Cat		
Yes	No	OR/95% CI
28	688	1
3	139	0.5/0.2 - 1.8
16	168	2.3/1.2 - 4.4
1*	24	1.0/0.1 - 7.8 (1.8)†
16	134	2.9/1.5 - 5.6
1*	25	1.0/0.1 - 7.5 (2.4)†

12. Angioni AM, Fanciulli G, Corchia C. Frequency of and risk factors for allergy in primary school children: results of a population survey. Paediatr Perinat Epidemiol 1989;3:248-55.
13. Pöysä L. Atopy in children with and without a family history of atopy. II. Skin reactivity. Acta Paediatr Scand 1989;78: 902-6.
14. Rugtveit J. Environmental factors in the first months of life and the possible relationship to later development of hypersensitivity. Allergy 1990;45:154-6.
15. Dowse GK, Turner KJ, Stewart GA, Alpers MP, Woolcock AJ. The association between Dermatophagoides mites and the increasing prevalence of asthma in village communities within the Papua New Guinea highlands. J ALLERGY CLIN IMMUNOL 1985;75:75-83.
16. Høst A, Husby S, Osterballe O. A prospective study of cow's milk allergy in exclusively breast-fed infants. Acta Paediatr Scand 1988;77:663-70.
17. Smith JM, Knowler LA. Epidemiology of asthma and allergic rhinitis. I. In a rural area. Am Rev Respir Dis 1965;92:16-30.
18. Kjellmann NIM. Immunoglobulin E and atopic allergy in childhood. Linkoping University Medical Dissertations, No. 36. Linkoping, Sweden, 1976.
19. Björksten F, Suoniemi I. Dependence of immediate hypersensitivity on the month of birth. Clin Allergy 1976;6:165.
20. Barbee RA, Lebowitz MD, Thompson HC, Burrows B. Immediate skin-test reactivity in a general population sample. Ann Intern Med 1976;84:129-33.
21. Linna O. Environmental and social influences on skin test results in children. Allergy 1983;38:513-6.
22. Smith JM. Epidemiology and natural history of asthma, allergic rhinitis and atopic dermatitis (eczema). In: Middleton E, Reed CE, Ellis EF, eds. Allergy, principles and practice, vol II. St. Louis: CV Mosby, 1988. 919.
23. Haahtela T, Björksten F, Heiskala M, Suoniemi I. Skin prick test reactivity to common allergens in Finnish adolescents. Allergy 1980;35:425-31.
24. Freidhoff LR, Meyers DA, Marsh DG. A genetic-epidemiologic study of human immune responsiveness to allergens in an industrial population II. The associations among skin sensitivity, total serum IgE, age, sex, and the reporting of allergies in a stratified random sample. J ALLERGY CLIN IMMUNOL 1984;73:490-9.
25. Gergen PJ, Turkeltaub PC, Kovar MG. The prevalence of allergic skin test reactivity to eight common aeroallergens in the U.S. population: results from the second National Health and Nutrition Examination Survey. J ALLERGY CLIN IMMUNOL 1987;80:669-79.
26. Arora S, Sanyal RK, West GB. The sensitizing action of Bordetella pertussis vaccine. Int Arch Allergy 1970;37:357-65.
27. Backman A, Björksten F, Ilmonen S, Juntunen K, Suoniemi I. Do infections in infancy affect sensitization to airborne allergens and development of atopic disease? Allergy 1984;39:309-15.
28. Mensinga TT, Schouten JP, Rijcken B, Weiss ST, Speizer FE, van der Lende R. The relationship of eosinophilia and positive skin test reactivity to respiratory symptom prevalence in a community-based population study. J ALLERGY CLIN IMMUNOL 1990;86:99-107.
29. Ronchetti R, Ciofetta G, Indinnimeo L, et al. Increased serum IgE and increased prevalence of eosinophilia in 9-year-old children of smoking parents. J ALLERGY CLIN IMMUNOL 1990;86:400-7.
30. Belin L, Dreborg S, Einarsson R, et al. Phazet—a new type of skin prick test. Calibration and stability. Allergy 1985;40: 60-3.
31. Harell FE. The practical value of logistic regression. In: Proceedings of the Tenth Annual SAS Users Group, International Conference. Cary, SAS Institute, 1985.
32. SAS Technical Report P-200. SAS/STAT Software: CALIS and LOGISTIC Procedures. Cary, SAS Institute, 1990.
33. Greenberg S, Kleinbaum DG. Mathematical modeling strategies for the analysis of epidemiologic research. Am Rev Public Health 1985;6:223-45.
34. Kleinbaum DG, Kupper LL, Morgenstern H. Epidemiologic research. Principles and quantitative methods. New York, Van Reinhold Nostrand A Company, 1982.
35. Strobel S, Ferguson. Immune responses to fed protein antigens in mice. Systemic tolerance or priming is related to age at which antigen is first encountered. Pediatr Res 1984;18:588-94.
36. Lucas A, Brooke OG, Morley R, Cole TJ, Bamford MF. Early diet of preterm infants and development of allergic or atopic disease: randomised prospective study. BMJ 1990;300:837-40.
37. Happle R, Schnyder UW. Evidence for the carter effect in atopy. Int Arch Allergy Appl Immunol 1982;68:90-92.
38. Åberg N, Engström I. Natural history of allergic diseases in children. Acta Paediatr Scand 1990;79:206-11.
39. Burney PG, Chinn S, Rona RJ. Has the prevalence of asthma increased in children? Evidence from the national study of health and growth 1973-86. BMJ 1990;300:1306-10.
40. Sears MR, Herbison GP, Holdaway MD, Hewitt CJ, Flannery EM, Silva PA. The relative risks of sensitivity to grass pollen, house dust mite and cat dander in the development of childhood asthma. Clin Exp Allergy 1989;19:419-24.
41. Croner S, Kjellman NIM. Development of atopic disease in relation to family history and cord blood IgE levels. Eleven-year follow-up in 1654 children. Pediatr Allergy Immunol 1989;2:14-20.
42. Brown EB, Josephson BM, Levine HS, Rosen M. A prospective study of allergy in a pediatric population. Am J Dis Child 1969;117:693-8.
43. Cederqvist LL, Eddey G, Abdel-Latif N, Litwin SD. The effect of smoking during pregnancy on cord blood and maternal serum immunoglobulin levels. Am J Obstet Gynecol 1984;148: 1123-6.
44. Magnusson CGM. Cord serum IgE in relation to family history and as predictor of atopic disease in early infancy. Allergy 1988;43:241-51.
45. Michel FB, Bousquet J, Greillier P, Robinet-Levy M, Coulomb Y. Comparison of cord blood immunoglobulin E concentrations and maternal allergy for the prediction of atopic diseases in infancy. J ALLERGY CLIN IMMUNOL 1980;65:422-30.

2024228033

Source: https://www.industrydocuments.ucsf.edu/docs/ppvj0000